Results The blood results of 440 patients were reviewed using the ICE (Integrated Clinical System). The median age was 67-years. Of the 440 patients, 310 had deranged LFTs, 86 had a normal liver profile, and 44 had no LFTs taken prior to ERCP. Of those with deranged LFTs, 2% had only a raised bilirubin, less than 6% had either only raised transaminases or both raised transaminases and bilirubin, 12% had only both raised alkaline phosphatase (ALP) and bilirubin, 15% had both raised ALP and transaminases, 22% had only raised ALP, and 43% had a trio of raised bilirubin, ALP, and transaminases. Only 11 patients had no coagulation profile taken before the procedure. Out of the remaining 429 patients, 15 were on warfarin, 2 had haemophilia, 1 was thrombocytopenic, and 1 had von Willebrand's disease. However, out of 440 patients only 2 had an INR greater than 1.5, 1 of them being on warfarin.

Conclusion These results suggest that routine measurement of coagulation profile is unnecessary. This would reduce time delays, decrease costs and avoid further tests in patients. We suggest that the current pre-ERCP investigation guidelines be reviewed.⁴

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Disclosure of Interest None Declared.

PTH-014 PANCREATIC DUCT DILATATION SHOULD BE INVESTIGATED WITH ENDOSCOPIC ULTRASOUND IF COMPUTERISED TOMOGRAPHY FAILS TO IDENTIFY A LESION

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Introduction A dilated Pancreatic Duct (PD) may be associated with pancreatic disease, but this is infrequently investigated further if no lesion is found on Computerised Tomography (CT). There is limited data on the role of Endoscopic Ultrasound (EUS) with PD dilatation without a cause on CT, as the literature mostly describes the utility of EUS with apparent pancreatic lesions. We describe our experience of EUS in identification of causative lesions not apparent on CT.

Methods Sixty-one (61) cases were identified and retrospectively reviewed between 2007-2013 by searching the EUS database at a university teaching hospital. All had CT +/- MRCP findings of dilated PD (+/- Common Bile Duct (CBD) dilatation) and no pancreatic lesion (54 patients) or oedema/unable to exclude a lesion (7).

Results Mean patient age was 70 (41-90). Indications for CT included abdominal pain 16; abnormal Liver Function Tests (LFT) 14 (3 jaundiced); weight loss 9; other 14 (eg. staging CT for lung ca, CT colonography for diarrhoea etc). Mean PD diameter was 6 mm (3-25 mm) and 30 had CBD dilatation. CT showed normal pancreatic parenchyma in 46 (76%); prominent ampulla 5 (8%); pancreatic cyst 5 (8%); calcification 4 (6.5%); pseudocyst 1 (1.5%).

After EUS, 49 (80%) had PD dilatation confirmed, whilst 31 (51%) also had CBD dilatation. 38 (62%) failed to identify a cause and hence agreed with CT. Of the remaining 23 (38%) there was disparity between CT and EUS. An FNA biopsy was performed in 16 (26%) of cases. Findings included neoplasm 9 (15%); IPMN 4 (6.5%); biliary stone disease 3 (5%); chronic pancreatitis 3 (5%); pseudocyst 1 (1.5%); choledochal cyst 1 (1.5%); and pancreas divisum 1 (1.5%). Neoplastic disease included pancreas cancer 5; suspicious ampullary tumour 2; cholangiocarcinoma 1; and mucinous cystadenoma 1.

With particular reference to EUS, there was isolated PD dilatation in 27 cases (44% of total), and abnormalities detected in (59%) which included cancer or IPMN (15%). In PD and CBD dilatation 22 (36%); 6 cases were abnormal of which 4 (18%) had cancer (pancreas and ampulla). Without PD dilatation 12 (20%), pathology was found in 50% including cholangiocarcinoma (1), IPMN (1), CBD stones (3), chronic pancreatitis (1). Of 9 cancer patients, dilatation was seen in PD only (4); PD and CBD (4); normal PD or CBD (1).

Conclusion PD dilatation should be investigated further with EUS, even when CT shows no causative lesion. We identified a significant percentage of benign (21%) and malignant (15%) pathology with EUS. EUS offers the additional advantage of biopsy when there is diagnostic doubt. Normal LFTs and the absence of the 'double duct sign' are insufficient to exclude neoplastic disease and EUS will help identify these.

Disclosure of Interest None Declared.

PTH-015 POST-ERCP PANCREATITIS IN SECONDARY CARE: CAN WE PREDICT WHO WOULD BENEFIT FROM A PROPHYLACTIC PANCREATIC STENT?

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Introduction Pancreatitis is a recognised complication of ERCP. Measures taken to reduce the incidence in high-risk patients include placement of prophylactic pancreatic stents and use of NSAIDs, but current practice varies widely. The frequency of post-ERCP pancreatitis (PEP) in unselected groups ranges from 1.3-6.7% in the largest reported series. Historically, prophylactic stents have not been used at our institution. This retrospective study was designed firstly to measure rates of PEP in our institution (a district general hospital), and secondly to identify those cases which may have benefitted from prophylactic pancreatic stenting.

Methods A retrospective database search identified all patients undergoing ERCP across our Trust over a 5-year period (April 2007 to July 2012). A linked search with a county-wide biochemistry database then identified all those patients who had subsequently developed an elevated serum amylase up to 7 days following ERCP. A consensus grading system was used to define PEP as: clinical pancreatitis (new/worsened abdominal pain), requiring or prolonging hospital admission by ≥ 2 days, with serum amylase over 3 times upper limit of normal >24 h post procedure (300 units/l). Additional risk factors for post-ERCP pancreatitis were identified: previous PEP, Sphincter of Oddi dysfunction, repeated cannulation, injection of contrast into pancreatic duct, Sphincter of Oddi manometry, balloon dilatation, precut sphincterotomy, or pancreatic sphincterotomy.

Results 2699 patients underwent ERCP during the study period. 6 patients were excluded due to incomplete records. 57 patients